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10/089,595	09/23/2002	Sunil Ahuja	4003.001600	2489

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EXAMINER	
SITTON, JEHANNE SOUAYA	
ART UNIT	PAPER NUMBER
1634	

SHORTENED STATUTORY PERIOD OF RESPONSE	MAIL DATE	DELIVERY MODE
3 MONTHS	01/19/2007	PAPER

Please find below and/or attached an Office communication concerning this application or proceeding.

If NO period for reply is specified above, the maximum statutory period will apply and will expire 6 MONTHS from the mailing date of this communication.

Office Action Summary	Application No.	Applicant(s)	
	10/089,595	AHUJA ET AL.	
	Examiner	Art Unit	
	Jehanne S. Sitton	1634	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *01 September 2006*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *56-58 and 63-113* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☒ Claim(s) *56 and 70* is/are allowed.

6)☒ Claim(s) *57,58,65 and 67-69, 71-113* is/are rejected.

7)☐ Claim(s) *63,64 and 66* is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date *9/2006*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

1. Currently, claims 56-58 and newly added claims 63-113 are pending in the instant application and under consideration at this time. The following rejections are newly applied, as necessitated by the amendments. They constitute the complete set being presently applied to the instant Application. This action is FINAL.

2. The text of those sections of Title 35, U.S. Code not included in this action can be found in a prior Office action.

3. The amendments to the specification have rendered the objection made at section 4 of the previous office action moot.

4. The claim objections made at section 6 of the previous office action are moot in view of the cancellation of the claims.

5. The rejection of claims 56-57 under 35 USC 102(b) over Mummidi made at section 10 of the previous office action is moot in view of the amendments to the claims to specifically require detection of the indicated haplotype pairs. Page 136 (lines 21-29) of the instant specification and figure 1D provide a definition for each of haplotypes HHA, HHB, HHC, HHD, HHE, HHF*1, HHF*2, HHG*1, and HHG*2. Mummidi does not teach or suggest any of the particular haplotype pairs recited in the claims, nor an association between the indicated haplotype pairs and risk of accelerated HIV-1 progression in any population, let alone in any specific racial or ethnic population.

6. The rejection of claim 56 as being anticipated by Kaslow is moot in view of the amendments to the claims, which are awarded benefit of priority to the '137 provisional

application. The subject matter of example 15 (relevant to claim 56), is not supported in the priority document of Kaslow, accordingly, the Kaslow patent is not prior art for claim 56.

7. The rejections under 35 USC 102(e) over Choi, and 102(a) over Gonzalez at sections 12-13 of the previous office action are moot in view of the amendments to the claims, which are awarded benefit of priority to the '137 provisional application.

8. The rejections made under 35 USC 103 at sections 16-17 of the previous office action are moot in view of the cancellation of the rejected claims.

Claim Rejections - 35 USC § 112

9. Claims 80-113 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

The independent claims 80, 87, 94-96, 101, 106 and 110 are indefinite in their recitation of "absence of a CCR5 haplotype pair". The independent claims are drawn to various methods of identifying wherein the presence of a particular haplotype pair identifies a subject as having an increased risk of accelerated HIV-1 disease progression or an increased risk of becoming infected with an HIV-1 virus. However, the claims ' active steps include detecting the absence of a particular haplotype pair, where neither the claim nor the specification make clear that detecting the absence of such haplotype pair provides for an increased risk as set forth in the preamble. Accordingly, the metes and bounds of the claim are unclear.

10. Claims 80-113 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention. This is a New Matter Rejection.

Claims 80, 87, 94-96, 101, 106 and 110 are drawn to identifying various human subjects as having an having an increased risk of accelerated HIV-1 disease progression or an increased risk of becoming infected with an HIV-1 virus by detecting the presence "or absence" of particular haplotypes. The response, at page 13-15, cites numerous portions of the specification for support for the instantly pending claims. Both the specification and the provisional application were thoroughly reviewed but do not appear to provide support methods of identifying subjects at risk by detecting the "absence" of a particular CCR5 haplotype. Additionally, newly added claims 80-93 broadly recite "identifying *a human subject* as having an increased risk" for having an increased risk of accelerated HIV-1 disease progression or an increased risk of becoming infected with an HIV-1 virus by detecting a particular CCR5 haplotype pair. Although the specification provides support for increased risk associated with particular haplotypes in specific racial populations, as in claims 56 and 57, the specification does not appear to provide support for the association in any human subject, regardless of race or ethnicity. The recitation in the newly added claims as set forth above appears to broaden the invention outside the scope of that which is supported by the specification and accordingly, have added new matter to the claimed invention.

11. Amended claim 58 and newly added claims 67-69, 71-74, and 76-113 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for methods of identifying Caucasian or African American human subjects as being at increased risk for accelerated HIV-1 disease progression comprising detecting the specific haplotype pairs set forth in claims 56 and 57 respectively, methods of identifying Caucasian human subjects as being at increased risk for infection with an HIV-1 virus comprising detecting the CCR5 HHE/HHE haplotype pair, as well as methods of identifying African American human subjects as being at increased risk for infection with an HIV-1 virus comprising detecting the CCR5 HHC/HHC haplotype pair, does not reasonably provide enablement for the methods recited in amended claim 58, or newly added claims 67-69, 71-74, and 76-113 for the reasons set forth below. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make or use the invention commensurate in scope with these claims. There are many factors to be considered when determining whether there is sufficient evidence to support determination that a disclosure does not satisfy the enablement requirements and whether any necessary experimentation is undue. These factors have been described by the court in *In re Wands*, 8 USPQ2d 1400 (CA FC 1988). *Wands* states at page 1404,

> "Factors to be considered in determining whether a disclosure would require undue experimentation have been summarized by the board in Ex parte Forman. They include (1) the quantity of experimentation necessary, (2) the amount of direction or guidance presented, (3) the presence or absence of working examples, (4) the nature of the invention, (5) the state of the prior art, (6) the relative skill of those in the art, (7) the predictability or unpredictability of the art, and (8) the breadth of the claims."

As the prior art teaches sequences which contain the 9 indicated positions for each of the haplotypes HHA-HHG*2 recited in Figure 1D, the prior is enabling for detecting or determining

the genotype of each of these positions. Further, although the specification is silent with regard to an association between increased risk of HIV-1 infection and the HHC/HHC haplotype in African Americans, the prior art of Kaslow (US patent 6,372,435) appears to be enabling for detecting increased risk of HIV-1 infection in African American human subjects by detecting the HHC/HHC haplotype pair (see table 4, P*0101/P*0101; table 7, col. 28, lines 1-9). The following rejection is being applied to the claims as they are drawn to identifying human subjects at increased risk for HIV-1 disease progression or of becoming infected with an HIV-1 virus based on detection of the recited haplotype pairs.

With regard to amended claim 58 and newly added claims 67-69; 72, 77-93 and 106-113, the claims are drawn to diagnostic methods of identifying any human child or any human subject, regardless of race, as being at increased risk of accelerated HIV-1 disease progression or at increased risk of becoming infected with an HIV-1 virus comprising detecting one of the specific haplotype pairs set forth in the cited claims. The specification at pages 135-142, teaches assessment of the CCR5 haplotype frequencies in mother to child HIV-1 transmission as well as HIV-1 disease progression for Argentinean children (see also figures 4A and 4B). However, the specification teaches that "The vast majority of Argentineans are descendants of individuals from Southern Europe, primarily from Spain and Italy. There is little admixture of Amerindians and there is no black population." (page 135). At pages 158-164 and figures 2 and 3, the specification teaches determining haplotype frequencies for Caucasians and African Americans, an teaches that in Caucasians and Hispanics HHC haplotypes were associated with disease retardation, particularly a delayed progression to death, while for African Americans, possession of HHC haplotypes was associated with disease acceleration. The specification also teaches that

HHE homozygosity, but no HHE heterozygosity was associated with disease acceleration in Caucasians, whereas this haplotype pair was not associated with disease modifying effects in African Americans. Additionally, the specification teaches "These findings also highlight the inter-racial heterogeneity of CCR5 resistance or susceptibility alleles and intra-locus allele interactions. Thus, genotype-phenotype association derived from one population *may not be generalizable to other populations*" (page 146, lines 14-15). Further, at page 145, the specification teaches "different pair wise combinations of CCR5 haplotypes may be associated with very different phenotypes, and the same haplotype pair may have different effects in different populations". Accordingly, although the specification is enabling for identifying a Caucasian human subject or an African American human subject as being at increased risk for accelerated HIV-1 disease progression by detecting the specific haplotype pairs in claims 56 and 57, respectively, the specification does not provide an enabling disclosure for identifying this risk or increased risk of becoming infected with an HIV-1 virus in any human subject or any human child, regardless of race. As evidenced by the teachings in the specification, there is no predictable association for increased risk of accelerated HIV-1 disease progression or increased risk of becoming infected with an HIV-1 virus between each specific haplotype pair and broadly "any" human subject. Therefore, the skilled artisan would be required to perform undue experimentation to make and use the invention as broadly as it is claimed. Given the conflicting teachings in the specification, as well as the lack of guidance in the art regarding the association of such haplotype pairs in any human population and increased risk of accelerated HIV-1 disease progression or HIV-1 infection, as is broadly claimed, the skilled artisan would be required to perform a large amount of unpredictable trial and error experimentation to determine whether the

specifically claimed haplotype pairs were in fact associated with increased risk of accelerated disease progression or infection in any human population.

With regard to claims 71, 73-74, 76, 87, 89, 90, 92, 101-103, and 105, the claims are drawn to methods of identifying African American human subjects at increased risk for HIV-1 infection by detecting one of the following specific haplotype pairs: HHC/HHF*1, HHC/HHE, or HHC/HHD. Although the specification teaches that these haplotype pairs are associated with accelerated HIV-1 disease progression in African American subjects (figure 3), the specification does not teach any working examples of identifying an increased risk of HIV-1 infection in African American subjects by detecting any of the haplotype pairs in claim 71, for example. The specification appears to base the claimed association for infection in African Americans on a study (example 6) which found that in Argentinean children specific haplotype pairs were found to be associated with both risk of infection and accelerated disease progression. However, it is noted that in Caucasians, while the HHE/HHE haplotype pair was found to be associated with increased risk of accelerated HIV-1 disease progression, the other two haplotype pairs found associated in Argentinean children: HHE/HHC and HHE/HHG*2 were not found to be similarly associated in adult Caucasians. Therefor, although the specification provides assessment of disease course in Argentinean children and Adult Hispanic Americans which found that homozygosity for HHE was associated with more rapid progression to death (page 142), no assessment is made with regard to adult African Americans. Further, Kaslow (US Patent 6,372,435), in providing an assessment of disease progression and risk of infection in Africans as well as a New York cohort, found that previously recognized relationships between two CCR5 promoter variants and HIV-1 disease progression almost contradicted their relationships to

infection (col. 35, lines 1-5). Accordingly, there is does not appear to be a predictable correlation between an increased risk of accelerated HIV-1 disease progression and increased risk of becoming infected with an HIV-1 virus by detecting the same haplotype pair. Therefore, although the level of skill in the art is high, the unpredictability is higher given the lack of guidance in the specification as to an association between the haplotype pairs HHC/HHF*1, HHC/HHE, or HHC/HHD and risk of HIV-1 infection in any human subject, or more specifically African Americans, as well as the conflicting teachings in the art regarding an association between CCR5 alleles and risk of HIV-1 disease progression vs risk of infection. The skilled artisan would be required to perform unpredictable trial and error experimentation to practice the invention as broadly as it is claimed.

With regard to claims 80-113, although the claims recite methods of identifying human subjects at increased risk of accelerated HIV-1 disease progression or infection, the claims also recite such diagnostics by detecting the "presence or absence" of particular haplotype pairs. Although the specification and prior art are enabling for the specific methods noted above, that is by detecting the presence of specific haplotype pairs, neither the claims nor the specification provide for identifying subjects at increased risk for disease progression or infection by detecting the absence of the specific haplotype pairs. Therefore, the skilled artisan would be required to perform undue experimentation to make and use the invention as broadly as it is claimed. Given the conflicting teachings in the specification, as well as the lack of guidance in the art regarding the association of the absence of such haplotype pairs in any human population and increased risk of accelerated HIV-1 disease progression or HIV-1 infection, as is broadly claimed, the skilled artisan would be required to perform a large amount of unpredictable trial and error

experimentation to determine whether the absence of the specifically claimed haplotype pairs were in fact associated with increased risk of accelerated disease progression or infection in any human population.

Claim Rejections - 35 USC § 102

12. Claims 80-105 are rejected under 35 U.S.C. 102(b) as being anticipated by Mummidi (Mummidi et al; Nature Medicine, vol. 4, July 1998, pages 786-793).

Claims 80-105 are drawn to detecting the presence "or absence" of particular haplotype pairs in humans. The claims only require detecting the presence or absence of particular haplotype pairs. At table 2, Mummidi teaches detection of the CCR5 Δ32 allele (deletion) in Caucasians. The specification at page 136 (lines 21-29) and figure 1D, define the 9 different CCR5 haplotypes (HHA, HHB, HHC, HHD, HHE, HHF*1, HHF*2, HHG*1, and HHG*2). The HHE haplotype is defined as including the CCR5 wt (or "+") allele (lack of 32 base pair deletion). Accordingly, Mummidi inherently teaches detecting the lack of a HHE/HHE haplotype pair in humans as well as human Caucasians because the HHE haplotype requires the CCR5 wt allele (lack of Δ32). Additionally, at table 2, Mummidi teaches detection of the CCR2 64I allele in Caucasians and African Americans. The specification at page 136, teaches that the only haplotype to contain the 64I variant is HHF*2. Accordingly, in teaching detection of the CCR2 64I variant, Mummidi inherently teaches detecting the absence of HHE/HHE, HHC/HHF*1, HHC/HHE, HHC/HHC, HHC/HHD, and HHE/HHG*2 in humans as well as human Caucasians and African Americans because each of these haplotypes require the CCR2 64V variant. It is noted that with regard to detecting "absence" of a particular haplotype pair, the

preamble has not been given any patentable weight as neither the claim nor the specification provide for identifying a human subject at risk by detecting the absence of the particular haplotype pairs recited.

13. Claims 57, 65, 71, 75, and 80-105 are rejected under 35 U.S.C. 102(e) as being anticipated by Kaslow (US Patent, 6,372,435).

With regard to claims 57, 65, 71, 75, 80, 84, 87, 91, 96, 99, 101, and 104, Kaslow correlates haplotypes with infection and HIV progression in different racial populations, including Africans (see Table 7, Table 9, Table 11; figure 3, col. 28). Kaslow teaches that the P*0101/P*0101 haplotype pair demonstrated association with HIV-1 infection and disease progression in Africans (see table 4, P*0101/P*0101; table 7, table 9, table 11, col. 28, lines 1-9). It is noted that the P*0101 haplotype taught by Kaslow corresponds to the HHC haplotype (see figure 6B) as it contains a G at position 59029 (corresponds to position 303 of the instant application), a T at position 59353 (627), a C at position 59356 (630), a G at position 59402 (position 676) and a C at position 59653 (927). As seen in figure 1D of the instant application, the only haplotype with the same alleles is HHC. Further, Kaslow teaches determining the same correlations in a New York cohort which included African Americans and teaches that Rwandans and black African Americans from New York closely resembled each other in promoter genotypic frequencies (col. 25, lines 60-64). As stated in the MPEP in chapter 2100:

Where the claimed and prior art products are identical or substantially identical in structure or composition, or are produced by identical or substantially identical processes, a prima facie case of either anticipation or obviousness has been established. In re Best, 562 F.2d 1252, 1255, 195 USPQ 430, 433 (CCPA 1977). "When the PTO shows a sound basis for believing that the products of the applicant and the prior art are the same, the applicant has the burden of showing that they are not." In re Spada, 911 F.2d 705, 709, 15 USPQ2d 1655, 1658 (Fed. Cir. 1990).

With regard to claims 85-105, which are also drawn to detecting the "absence" of particular haplotype pairs in humans. At table 5, Kaslow teaches detection of the CCR5 Δ32 allele (deletion) in Caucasians (annotation b). The specification at page 136 (lines 21-29) and figure 1D, define the 9 different CCR5 haplotypes (HHA, HHB, HHC, HHD, HHE, HHF*1, HHF*2, HHG*1, and HHG*2). The HHE haplotype is defined as including the CCR5 wt (or "+") allele (lack of 32 base pair deletion). Accordingly, Kaslow inherently teaches detecting the lack of a HHE/HHE haplotype pair in humans as well as human Caucasians because the HHE haplotype requires the CCR5 wt allele (lack of Δ32). Additionally, at table 8, Kaslow teaches detection of the CCR2 64I allele in Caucasians and African Americans. The specification at page 136, teaches that the only haplotype to contain the 64I variant is HHF*2. Accordingly, in teaching detection of the CCR2 64I variant, Kaslow inherently teaches detecting the absence of HHE/HHE, HHC/HHF*1, HHC/HHE, HHC/HHC, HHC/HHD, and HHE/HHG*2 in humans as well as human Caucasians and African Americans because each of these haplotypes require the CCR2 64V variant. It is noted that with regard to detecting "absence" of a particular haplotype pair, the preamble has not been given any patentable weight as neither the claim nor the specification provide for identifying a human subject at risk by detecting the absence of the particular haplotype pairs recited.

The specific haplotype pairs HHE/HHE, HHC/HHF*1, HHC/HHE, and HHC/HHD are not taught by Kaslow as the genotype assessments made by Kaslow do not distinguish HHE from HHG*1 from HHG*2 (P*0201), or HHF*1 from HHF*2 (P*0202). Further, Kaslow teaches that the haplotype pair HHC/HHD (P*0101/*0103) was not found (col. 28, lines 51-52).

14. Claims 106-108, and 110-112 are rejected under 35 USC 102(b) as being anticipated by Buseyne (Buseyne et al; Journal of Infectious Diseases, October 1998, vol. 178, pages 1019-1023).

Claims 106-108 and 110-112 are drawn to detecting the presence "or absence" of particular haplotype pairs in a human child. The claims only require detecting the presence or absence of particular haplotype pairs.

Buseyne teaches determining the CCR5 32 base pair deletion in perinatally HIV infected children and correlating the impact of heterozygosity for the receptor with plasma viral load and CD4 T lymphocytes (see abstract). The specification at page 136 teaches that the only haplotype to contain the CCR5 Δ32 allele is HHG*2. Accordingly, in teaching detection of CCR5 Δ32 allele, Buseyne inherently teaches detecting the absence of HHE/HHE, and HHC/HHE as these haplotype pairs contain the CCR5 "+" or "wt" allele (lack of CCR5 Δ32 allele).

It is noted that with regard to detecting "absence" of a particular haplotype pair, the preamble has not been given any patentable weight as neither the claim nor the specification provide for identifying a human subject at risk by detecting the absence of the particular haplotype pairs recited.

15. Claims 106-113 are rejected under 35 USC 102(a) as being anticipated by Szalai (Szalai et al; Pediatric Research, July 1999; Vol 46, abstract).

Claims 106-113 are drawn to detecting the presence "or absence" of particular haplotype pairs in a human child. The claims do not require actually detecting the identity of the particular 9 genotype positions of the recited haplotypes, they only require detecting the presence or

absence of particular haplotype pairs. Szalai teaches detecting the CCR2 64I variant in children. The specification at page 136, teaches that the only haplotype to contain the 64I variant is HHF*2. Accordingly, in teaching detection of the CCR2 64I variant, Szalai inherently teaches detecting the absence of HHC/HHE, HHE/HHE, and HHE/HHG*2 because these haplotype pairs contain the CCR2 64V variant.

It is noted that with regard to detecting "absence" of a particular haplotype pair, the preamble has not been given any patentable weight as neither the claim nor the specification provide for identifying a human subject at risk by detecting the absence of the particular haplotype pairs recited.

Conclusion

16. Claims 56 and 70 are allowable over the cited prior art.

17. Claims 63, 64, and 66 are objected to for being dependent on a rejected claim.

18. Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event,

however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

19. Any inquiry concerning this communication or earlier communications from the examiner should be directed to examiner Jehanne Sitton whose telephone number is (571) 272-0752. The examiner can normally be reached Monday-Thursday from 8:00 AM to 5:00 PM and on alternate Fridays.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Ram Shukla, can be reached on (571) 272-0735. The fax phone number for this Group is (571) 273-8300.

Any inquiry of a general nature or relating to the status of this application or proceeding should be directed to (571) 272-0547.

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11/27/06